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THE EFFECT OF DAMAGE TO OPTICAL PATHWAYS ON POSTURE AND MOVEMENT[1]

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Of all man's senses, his ability to see is undoubtedly the most important. It is through vision that we derive pleasure from television, movies and ballet, that we can enjoy the convenience of driving a motor vehicle and maintain the ability to protect ourselves from other lethal weapons on our roads. Memory, dreams, imagination, sound, smell and taste all have visual associations. We judge time, in seconds, days or seasons by what we see. Memory in particular is based to a large extent on visual experiences, and those who lose vision can still recall its highlights and understand its meaning. Those who are born blind must have great difficulty in understanding seemingly simple things we take for granted, such as the meaning of light and dark, the concepts of colour, the beauty of sunsets, and the information imparted by various facial expressions. We with vision can observe a small degree of contraction of the orbicularis oris which changes an insulting remark into a humorous one.

Reflecting the importance of vision, the human brain is connected intricately with, and dominated in part by the visual pathways, from the eyes in the front through to the occipital cortex at the back, and as well, up and down the brain stem.

The retina is the seeing portion of the eye that transmits a light stimulus into a neuronal excitation into the optic nerve. Even before it reaches the retina, vision can be interrupted by such things as a scar on the cornea or a cataract of the lens. The retina itself is prone to detachment, inflammation, and degeneration. The optic nerve consists of myelinated nerve fibres and meningeal sheaths, extending from the globe anteriorly to the chiasm posteriorly, and it can be traumatised, compressed by tumours, and damaged by demyelination. Lesions of any of these structures produce what is called monocular defects, which may be total or which may affect a segment or segments of the visual field of that eye. Some of these diseases may involve both eyes, when naturally, the visual acuity will be more severely impaired. However, no direct or immediate effect on posture or movement is seen with lesions at these sites. Both eyes will move synchronously and people with monocular blindness, though losing some degree of depth perception, can continue, without apparent ill effect, to carry out highly developed motor skills, as exemplified by the numerous people with one eye who have been very prominent in sport.

At the optic chiasm, the nerve fibres separate, according to the rule for intracranial lateralisation of sensory and motor representations; the fibres from the medial portions of the optic nerve, which represent the lateral visual fields, cross over to the contralateral geniculate body (and midbrain), while fibres from the lateral portions of the nerve, representing the medial visual fields, remain uncrossed to terminate in the homolateral geniculate body. These fibres going from the chiasm to the lateral geniculate bodies are called the optic tracts.

The chiasm can be damaged by tumors in and around the pituitary gland, and the effect on vision with chiasmatic lesions is often a bitemporal field defect, that is, outer or lateral

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vision of both eyes is affected. This may affect our visual protective reflexes, but again no specific change in posture or movement is observed.

With lesions of the optic tract, yet again a different type of visual field defect is produced, namely an hemianopia, where either the right or the left field of vision is involved, rather than an individual eye. Being close to the diencephalon, midbrain and cerebral peduncles, lesions of the optic tracts are sometimes accompanied by other signs, including pyramidal weakness on the side of the field loss.

The geniculo-calcarine radiations fan out to circumvent the lateral ventricles on their way to the occipital cortex, the lower fibres swinging around the temporal horns in the temporal lobes, while the upper fibres pass directly through the parietal lobes. It is in the parietal lobes especially that one might see some strange disorders of movement.

The parietal lobes are the principal sensory areas of the cerebral cortex, the parts of the body being represented in a similar manner to the motor system, with the leg being superior and the head inferior. Cortical sensory loss does not significantly affect pain, thermal sensation, light touch or vibration sense; rather it is one's ability to tell the size, shape, texture and weight of objects placed in the hand, which involves stereognosis, joint position sense and the localisation of sensory stimuli. In the absence of these modalities, visual, and to a lesser degree, labyrinthine functions help to overcome the deficit. Lesions of the visual pathways in a parietal lobe will lead to hemianopia, often incomplete and only in the lower field if the parietal lobe alone is involved. Consequently, enough vision is usually left to aid in compensation for any associated sensory loss. Naturally it is possible for bilateral lesions to occur, but in these rare instances the loss of higher functions is so devastating that discussion on the effects on posture and movement is of little practical importance.

Lesions of the visual pathways as they pass through the parietal lobes may lead to hypotonia, wasting and static as well as kinetic ataxia. The affected arm may be restless, sometimes amounting to pseudo-athetosis. Gesticulations may be exaggerated and when the arm is held in front of the body with the eyes closed, it may wander up and away. Other disturbances may also occur which may have an indirect effect on posture and movement. Loss of colour perception, loss of ability to calculate, confusion between left and right, failure to recognise one's individual fingers, and failure to recognise common objects and even well-known faces (presopagnosia) can all occur with lesions in this vicinity. Very important particularly is apraxia, where a patient cannot carry out a purposive movement, even though he understands the command, and has no motor, sensory or cerebellar deficit. To the uninitiated, an apraxia can be mistaken for dementia, poor co-operation, cussedness or hysteria. Apraxia may involve such fundamental movements as sitting and walking, which if it occurs alone may present a striking and sometimes diagnostically confusing picture, as will be seen in the following case report:

Case 1

A tank commander, aged 28, was struck in the back of the head by a shell fragment. He sustained a penetrating wound of the occiput from which brain was protruding when he was first seen. He was found to be responding to questions. His only complaint was blindness. When asked to move his right or left leg, he moved either arm indifferently and insisted he was moving his legs. He was seen by a neurologist 60 hours after wounding. He was then moderately confused. He was then able to see, though he was very hazy in the lower half of each visual field. The only other abnormality was that he would not or could not move the correct limb on command, and this was out of proportion to the degree of confusion. X-ray examination of the skull showed a midline high occipital bone defect with radiating fissures and indriven bone fragments to a depth of 4 cm.

Once he emerged from his confusion, he showed a difficulty with visual imagery and visual recall. He would picture a uniform and try to attach the rank badges without finding the proper place for them. As he said himself a little later, "I knew I had two pips, but I couldn't see where I could wear them — wherever I pictured them, they seemed odd and absurd. It all came back to me quite suddenly, and I felt how stupid I had been ever to be uncertain." By the end of that first month his visual fields had improved and he then had only a homonymous defect of the right inferior quadrants. This remained a permanent defect.

He was then allowed up, and at once became aware of a queer inability to place himself correctly when getting into bed or trying to sit down. On his second day up, he went into the lavatory and found

he could not work out which way he must face in order to end up in the conventional position. He began by bending his knees and found them on the seat. This went on until he anchored himself by placing his hands on one of the side walls and keeping them there while bending the knees. This enabled him to sit sideways, which was a satisfactory compromise to begin with, though he soon shuffled around to the front. As this happened on several days he tentatively mentioned it during a ward visit. Like so many patients with these bizarre disorders, he was afraid that it meant insanity. When reassured he talked freely about this and applied himself to overcoming the defect. He soon worked out a technique of holding on to the lavatory door handle instead of the side wall, before bending his knees.

Two months after the wounding he discussed his difficulty in getting into bed. If he was sitting on the side of the bed and was told to get in, as often as not he would end up with his feet on the pillows and his head at the foot. He would get out to rectify this and often repeat the error. The same thing would happen if he told himself to get into bed, whereas if while undressing he started talking to another patient, then he would get into bed without thinking about it and find himself the right way up. One day when asked to get into bed to be examined, he was obviously in great difficulty, and helped himself by head and tongue movements, such as may be seen in a child trying to solve a difficult problem. When asked what the difficulty was, he said, "Once I take my eyes off the bed I'm lost, because I can't picture myself and the bed at the same time". It was certainly true that if he faced the bed and climbed in, he achieved the correct ending more often than when sitting on the side. No defect of visual imagery for pictures, objects, or topography could be found at this time.

The following episode disturbed him greatly because it illustrated his difficulty in bodily orientation.

Because of the scar on his occiput he had become sensitive about the appearance of the back of his head. In places where he took off his hat, he liked to sit with his back to the wall. In a favourite restaurant frequented by his regiment there were only round tables with as many as a dozen chairs. He could never decide which chair to sit on in order to have the back of his head towards the wall. As a rule he gave up trying to work this out, but sat on any chair and moved round until he got into the right place.

Another interesting defect this patient showed was a difficulty in rapid visual interpretation. He had no visual agnosia as ordinarily interested, but in the cinema he found that he could follow only slowly-paced action. Any rapidly changing scene or much activity on the screen was completely beyond him. After a short time he would abandon the effort of following visually and hope the accompanying sound would keep him in touch until he could once more see what was happening. Like other patients with this disorder he preferred the theatre, where as a rule most of the action is static, to the cinema.

This patient was seen again a year later and improvement had continued. The visual fields were unchanged. Now he could usually put himself into a desired chair, though on important occasions, or where fear of failure or ridicule was present, he would resort to trial and error. He had no trouble with lavatory seats, though occasionally his knees would begin to bend too soon, but this he could control. He could now manage the cinema. He had no apraxia, constructional or otherwise, and no defect of the body scheme as applied to himself or the examiner. Formal intelligence testing gave an I.Q. of 120 on the Wechsler battery of tests.

This patient was described by Dr. Michael Kremer in his Oliver Sharpey Lectures delivered before the Royal College of Physicians of London in February, 1958. Although the defect of movement is unusual, it demonstrates extremely well what complex disorders of movement can occur with lesions involving the optic pathways and adjacent structures.

Involvement of the visual pathways as they traverse the non-dominant parietal lobe will produce a left hemianopia. Some of the associated disturbances include autotopagnosia which is unawareness or even denial of the left side of the body, anosognosia which is denial of disease on that side, even in the presence of a dense hemiplegia, a topographical agnosia where the patient cannot recognise what had been familiar territory and local geography so that he may lose his way even around his own home, and a dressing apraxia in which even the simple act of putting on glasses may prove to be an insurmountable task.

Apart from visual loss, other visual symptoms may occur with lesions of the geniculocalcarine pathways, including a deficiency in stereopsis or judging distances, teleopsia, where objects seem far away as though looking through the wrong end of binoculars, micropsia, where objects look falsely small, and visual hallucinations, both formed and unformed. These visual disturbances may affect the confidence of a patient, which in turn might make movement hesitant and slow.

Lesions of the optic radiations as they pass through the temporal lobes are not accompanied by such a variety of fascinating and discernible neurological defects as one sees with parietal lesions. Speech may be affected if the dominant temporal lobe is involved, but the other parameters of temporal lobe function such as auditory and visual perceptions, learning, memory and emotional reac-

tions are seemingly unaffected except in massive or bilateral lesions. This is why a right temporal lobe lesion may be accompanied only by a left superior quadrantic hemianopia, frequently ignored or unnoticed by the patient and not accompanied by any obvious disturbances of movement.

The occipital lobes are the terminus of the visual pathways, each lobe being divided into three areas, designated by the numbers 17, 18 and 19. Area 17 is concerned with reception of the primary visual stimulus. Area 18 synthesizes visual impressions received in area 17 and has abundant commissure connections with the corresponding occipital lobe but rather limited connections elsewhere. Area 19 on the other hand is involved with the rest of the brain and integrates visual impressions with speech, hearing and other functions of the sensorium as well as serving for visual memory. In addition to the cerebral cortical connections, efferent links also occur with the thalami, superior colliculi and pons. It is these connections which integrate our visual experiences, past and immediate with the rest of the brain, and especially the pyramidal, extrapyramidal and cerebellar systems. Thus, the most fascinating and greatest computer of all, the human brain, can protect and guide the body which it so elegantly controls, and it can be seen that a large part of the input into this computer is by way of the visual system.

Lesions of both occipital cortices do occur in clinical practice, because of their anatomical vulnerability to trauma, as well as their blood supply which is derived from one solitary artery, the basilar, which if occluded will affect both right and left posterior cerebral arterial supply. Patients with occipital blindness may deny their defect, which can be quite confusing, especially as the pupils will react to light because this particular reflex happens to bypass the occipital cortex.

Case 2

In January, 1968, a 54-year-old hotelkeeper was found by his wife crawling around on his hands and knees. He was put to bed. The next day he asked that the light be turned on, but he did not complain of blindness.

Initially his wife thought he was drunk, but when he continued to act strangely, she consulted their local medical practitioner.

When I first saw him in hospital he did not complain about his vision. When specifically questioned he admitted that he could not see well, but denied he was blind.

Examination revealed that his pupils reacted to light, yet he could not perceive light in either eye. His recent memory was poor, and he had a right-sided cerebellar ataxia. He could walk, though he veered to the right. There were no other neurological signs.

Originally it was considered that the cause of his blindness was due to bilateral optic neuritis, but the fact that his pupils reacted to light yet he could not see, his denial of blindness, as well as the history and other signs, suggested that he had occipital blindness. This was confirmed by vertebral arteriography, when a paucity of parietal and occipital branches of the posterior cerebral arteries was seen. Subsequently a minute tunnel of vision has returned. His right cerebellar signs have almost resolved.

This case is of interest for several reasons. First, it shows that blindness may be denied in certain rare circumstances. Secondly, it shows that the cause of blindness is not always easy to discern. The disturbance of movement (right cerebellar dysfunction) was due to a separate lesion, presumably an infarct in the right brain stem or cerebellum, and only related to his blindness in that both lesions were due to the same pathological process, namely infarction. The fact that he was found initially crawling around on his hands and knees, was probably due to a combination of the ataxia and the sudden loss of vision.

It is via the integration of our visual system that we can negotiate, for example, the highways. A sudden stimulus in the periphery of one field might lead to a turning of the head and eyes towards it, the movement of braking with one foot, pressing in the clutch with the other, protecting the forward lurch of a child with one hand and manipulation of the steering wheel with the other. Movements which we make are often reflex — either protective or emotional. We duck as our car passes under a low branch of a tree, even though we have a roof above us; we flinch or jump if someone feigns to strike us; we blink with visual threat, and if you observe spectators at a boxing contest or football match, you will witness reflex punching and kicking at appropriate moments. Because of the messages integrated and distributed from our visual cortices, we are able to get about — rapidly, safely and directly, without fear of tripping over or falling into something

beneath us, without fear of bumping into something around us or striking our heads on something above us.

In our every-day activities, which involve movement and posture, such as sitting, standing, walking, feeding, writing and performing various manual tasks, we rely on a complex integration of many impulses. Take for example our ability to stand. There we rely, amongst other things, on proprioceptive impulses arising from joints and muscles in our legs and trunk, as well as labyrinthine and visual stimuli. Frequently the benefit of vision is removed naturally, such as when we stand out of bed on a dark night. That we don't fall over reflects the great powers of our labyrinthine and proprioceptive reflexes. Likewise, when proprioceptive impulses are impaired, for example with certain lesions of the spinal cord, then vision becomes all important. This is exemplified by observing someone with, say, tabes dorsalis as he washes his face. Because proprioception from the legs is lost, as soon as the eyes are closed he falls into the basin, for then only labyrinthine reflexes are functioning.

It can be seen then that partial lesions of the visual pathways, whether they be at any site in the optical pathways, if pure, will not noticeably affect movement or posture. With complete lesions, all movements are possible — we all move normally in total darkness every night. Naturally we are more tentative if our visual memory is not good, and when we are in a strange environment we use our arms for additional tactile and proprioceptive information. A good example of the part vision plays in our judgement of distance, speed and movement is when one is blindfolded and lifted on a chair. A person then has no way of judging height and this can be a terrifying experience, producing quite grotesque postures and movements because of fear and lack of confidence.

Visual memory plays an enormous part in all our learning processes, including all the simple and complicated movements we perform. Yet we can do without it and some of the highest motor tasks, such as piano playing, have been carried out by people born blind. Their auditory, tactile and proprioceptive functions must be remarkable.

To complete this discussion, mention must be made of the oculomotor system—the third, fourth and sixth cranial nerves. The 3rd and 4th nuclei lie in the midbrain, while the 6th is in the pons. Together with the midbrain nuclei for pupillary light reaction and convergence, the oculomotor nerves move the eyes laterally, medially, up and down, and rotate them as well as constricting and dilating the pupils. The supranuclear pathways of these muscles are all important for they are connected not only amongst themselves, but also to other parts of the brain. With lesions of the oculomotor nerves and their connections, one does see more obvious effects on posture and movement, although usually confined to the eyes and head. Lower motor lesions will produce partial or complete paralysis of one or more ocular muscles, the 4th and 6th nerves supplying one muscle each, while the 3rd supplies several. Diplopia resulting from a paralysis of one of these nerves may be accompanied by head tilt and when acute and early, lack of confidence and co-ordination may ensue. Eventually, however, one eye is suppressed, either physically or subconsciously and no gross general effect on posture and movement will then be seen.

The supranuclear connections of the oculomotor nerves are many and complex. An area for conjugate deviation of the eyes exists in the frontal and occipital lobes on each side, which if stimulated will cause the head and eyes to deviate towards the opposite side. The ability of the eyes to look up and down also has a higher control, probably in the region of the periaqueduct grey matter. Another supranuclear pathway is the medial longitudinal fasciculus which extends caudally as far as the cervical cord. There are also important connections with the vestibular system.

Reflex movements of the eyes occur normally with retinal, auditory and labyrinthine stimuli — we hear and then look.

Lesions involving the supranuclear pathways result in paralysis or distortions of ocular movements. Paralysis of gaze in one direction can occur. The clinician may see nystagmus, which is a disturbance of ocular posture characterised by a more or less rhythmical oscillation of the eyes. This can occur in early life with severe impairment of

vision, and in later life with miners who work in poor light. Labyrinthine disorders will produce nystagmus, and we make use of this reflex in caloric testing, when we stimulate the labyrinth with hot or cold water run into the ears. The duration of nystagmus which follows provides useful clinical information.

Lesions in the midbrain, pons and brain stem may all be accompanied by bizarre disturbances of ocular posture and movement, including convergence spasms, see-saw nystagmus, and ataxic nystagmus, to name just a few.

SUMMARY

I have endeavoured to outline the vast and complex connections which our visual system has with the rest of the brain. As a general rule, gross disturbances of movement and posture are not seen with partial or incomplete lesions of this system. With lesions of the optic pathways as they traverse the parietal lobes, one may see associated disturbances of motor function, such as the apraxias. With lesions of the oculomotor system, various types of disturbance of ocular posture and movement can occur, which are usually confined to movements of the eyes only.

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